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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Cerebral Traumata — Concept and Valuation of Psychophysiologic Aftermats

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New York, N. Y.

Connection between head trauma and the appearance of mental and physical disorder often presents a complex matter both for decision, and for that which flows therefrom. This is especially so because of the frequency of medico-legal implications and the possibility of attendant complications. The automobile age, the now well recognized industrial hazards with the consequent protective legislation better known as the Workmen's Compensation law, the perils of warfare, the matured airplane era, as well as the exposures incident to athletic participation, and the occurrences peculiar to every day living, are all situations to conjure with, when endeavoring to identify and to appraise psychophysiologic disorders as they may reflect cranio-cerebral traumatic causality.

When such disorders appear subsequent to head trauma, causal relation to the latter is prone to be ascribed and affixed thereto by the laity wherever possible. In the first place, to non-medical people, a blow or other injury to the head however sustained, becomes a seemingly logical reason for the appearance of mental symptoms; and moreover (based on this reasoning) provided a motivating head trauma can be implicated, the involvement of the strain of biologic transmission of heritable genetic characters to the traumatized individual from the latter's parents is thereby avoided.

Injuries to brain tissue can occur in various ways. From mechanical forces applied to the cranial vault, I have seen casualties following falls from stairways, falls out of windows, after automobile mishaps, and those resulting from altercations where fists or other objects were forcibly applied to the victim's head. As a result of other causes, I have seen such cerebral injuries as those attending exposure to high tension electrical charges, skull penetration by missiles reaching brain substance, blast explosions where no outward evidence of injury is visible, etc. . . .

The results are manifold and their severity and chronicity depend upon the extent of the trauma as well as the locality of the tissue implicated. In my confirmed and extended personal observations of the varying gradations of psychological aberrations, as carried out in my services in hospital wards, out patient clinics, as well as other consultative settings, all seen in their respective surroundings, I have found that relatively fewer than 1% have demonstrated permanence of psychotic disorder following cerebral impact (1).

I have been struck by the frequency of correlative findings between the nature and severity of the mental syndromic picture met with in patients after head traumata, and the disclosures in their clinical histories respecting their personality liabilities antedating their accidents.

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According to their similarity of symptoms, signs, and course, but manifestly not probative of the precise lesions in the absence of surgery or necropsy, elemental factors determinative of brain injury may be structurally set apart under the pathological groupings of (a) concussion—(b) contusion—(c) laceration—and (d) hemorrhage of the brain. Consideration of their several cardinal pathological entities is presented to indicate the distinguishing characteristics of each of the component subheads antecedent to their individual interpretative discussion.

(a) *Concussion,* produced by a blow to the head is a disseminated rather than a focal brain disorder. Mental awareness to surroundings comes to an end, though the lower nerve centers continue their functional activities uncontrolled by those at cephalic levels. Thus, as a disturbance of function many explanations of its mechanism have been propounded. I mention the neuronal breakdown of polarized cell membranes with axon discharge; nerve cell displacement with synaptic disruption; creation of a disturbance through the brain fluids, arachnoid spaces and ventricles; cortical cessation of activity with unconsciousness resulting from sudden and extensive electrical potential release—all of which bespeak the brief excitatory space of time during which the concussed individual is "seeing stars" prior to unconsciousness.[2,3] The kinetics of concussion may be placed upon a sound physiological foundation on the basis of the animal experimental work of Walker and his associates.[4] The latters' following clinical recordations should clarify our understanding: At the moment of the blow applied to the head (concussion), cortical activity becomes increasingly frequent and occurs as an electrical discharge within the central nervous system. The activity follows for 10 to 20 seconds, is then decreased until little spontaneous discharge remains, and within several minutes the encephalographic picture shows practical normality again.

(b) *Contusion* varies in its relative severity from concussion, on the basis of the extent of the force applied to the skull and the latter's resistance to the blow. If the force is high and the skull's resistance is very low, cerebral contusion invariably takes place. Its pathogenesis ranges from: increased fluid formation and capillary bleeding; dotted hemorrhages and swelling-out of spaces around blood vessels and cells; localized demyelination[5]; to cavitation within the choroid plexus and cell structures lining the ventricles. The picture becomes further complicated when inflammatory meninges adhere and obstruct passage of spinal fluid. In this situation arachnoid tearing may follow and stimulate subdural cyst formations containing spinal fluid.

(c) *Laceration* of brain substance may take place either at the site of the applied force to the skull or the lesion may occur at a distant point. When the brain laceration takes place at a site remote from the region of impact the pathogenesis may be best understood by Henri Duret's theory that a cone of depression of spinal fluid is formed at the location directly struck, and a compensatory cone of elevation is created in the opposite region, causing forcible dissolution of brain integrity thereat.

(d) *Hemorrhage* of the brain, both extra—and subdural, are productive of hematomata. The latter, when of the gradually developing type, expand and thereby reduce the volume of brain substance. Subdural hematomata occur more frequently and are usually found within the skull opposite to the point on the skull's surface where the blow was applied. This physical mechanism may be explained under the theory of Duret, already referred to in the preceding subdivision, or the postulate identified as a "contre coupe." The latter comprehends the physical concept that the brain moves with the cranium and when the movement is suddenly halted the brain swings against the opposite side of the skull with a force equal to that of the external blow.[2] When of the subdural type, hemorrhages within the latter envelope are resultants of severe lacerations of the cerebral cortex with hemorrhagic inundation frequently reaching directly into the brain substance. When extradural, the bleeding most often follows skull fracture with the bone fragments tearing into the middle meningeal artery.

Significant Diagnostic Landmarks and Their Related Prognostic Estimation

Cerebral *concussion,* in the words of Russell, follows a cerebral mechanical agitation[6] without its bruising, laceration or puncture. As this is a definite trauma, causative of reversible as well as irreversible disturbances, the term should really be included as a part of the descriptive nomenclature of every brain injury. As postulated by Trotter[7] the shaking up of the boxed-in brain inside the skull should not presuppose "some mysterious though happily transient detriment thereby." Nevertheless the term has become a permanently attached diagnostic title for a condition which follows a blow to the head with or without a variable amount of disturbed consciousness, frequent brief periods of altered bodily functioning, but absence of neurological symptoms expressive of structural cerebral change. Following a head trauma, if the unconscious episode is short-lived and no after effects supervene, the patient, none the worse for his experience, usually seeks no medical assistance. Those patients who reach hospitalization and show no evidence of developing brain injury, should be continued under some limited hospital scrutiny. I have been guided in this class of cases on my service, by an adopted procedure not to sanction medical release of any hospitalized case, no matter how trivial the history of head trauma—unless the patient has had a complete neurological work-up with lumbar tap in indicated cases—until the patient has been observed for at least a minimum period of 72 hours from time of admission, with further stay contingent upon any evidence of signs of developing psychic variants.

As the manner of treatment is not the subject of this study it will not be developed here.

Where it had existed at time of trauma, the cardinal diagnostic landmarks of this category of cerebral impact, are the brief absence of awareness of one's own existence, with absence of demonstrable neurological findings. It should be emphasized however that an unconscious episode after head trauma is not universally present. Permanent amnesia for the occurrence of the event just preceding skull impact and for the intervening unconscious period always remains, once unconsciousness had occurred at injury. "The amnesia thus experienced by one who has suffered a head concussion, whenever such trauma is followed by a variable interval of unconsciousness, may be of two types: (a) First, the patient is unable to remember the specific experience and time when the injury took place, and at times will also remain amnestic for an indeterminate period prior to the traumatic event. It is to be borne in mind, also, that after recovery from the blow to the skull the actual traumatic episode is never recalled once the consciousness has been lost, irrespective of the length of the period of this unconsciousness. If, in addition, loss of memory also exists for events which took place immediately before the blow to the skull occurred, such amnesia indicates that the higher integrative cortical functions have suffered as a result of the trauma. This want of recall has been expressed as *retrograde amnesia.* (b) If, on return to consciousness, the traumatized patient appears bewildered or experiences a blunting of awareness, the latter will invariably be followed by a blotting out of the memory for such bewildered or clouded period. This memory gap has been variously expressed as *anterograde* or *post traumatic amnesia.*"[8]

I am entirel yin accord with Symonds[9] that it is doubtful whether persistent mental dysfunction can result from injury such as I have here detailed under the subdivision of cerebral concussion—mild type of head injury without demonstrable neurological signs and symptoms. Although irreversible structural manifestations may not follow concussion, I have frequently observed sundry psychic manifestations as aftermaths thereof.

Thus, in uncomplicated concussion, the practically uniform symptom is unconsciousness for variable periods, together with the amnestic experiences already detailed. As the consciousness returns, these patients exhibit their most usual complaint of headache of varying degree and variously localized, bouts of vertigo (positional or rotational) may follow, with vomiting being fairly common for the first day or two after the consciousness has returned.

As previously pointed out elsewhere by the writer,[3] as detailed an account as can be obtained from the patient, his associates, friends and relatives, should be gathered covering the patient's adaptability, initiative, aspirations and intentions present before the advent of his head trauma. This, in order to evaluate as far as possible, the patient's personality make-up, pre-traumatic psychic status; and to recognize any current or presently existing alteration in his surrounding circumstances, presence of adverse environmental situations, anxiety as to future work procurement, and for compensatory expectations since the injury.

Generally speaking, where neuroses are demonstrable as an aftermath of concussion, these neurotic changes demonstrate a difference in a quality from neuroses which may be the resultants of other bodily accidents where the force was not applied directly to the head. It has seemed to me that, in observing those who have been subjected to head trauma, I have found that they quite invariably adopt a peculiar motivation and evaluation in respect of the quality of their accident because it occurred to this particular section of the body—the head. It is well to bear in mind that medical consideration should be given to the question not only whether the traumatized individual will be able to resume his work again, but whether or not he suffers psychologically as a result of this injury. In my observations (particularly in early hospitalized cases of so-called simple concussion) I have noted that where unconsciousness had taken place for whatever period, sundry qualities of disorientation as to time, location, image, situation, or any combination of them, will follow. These patients may also exhibit phases of defective thinking and perception. I have seen them in euphoric states where they minimize their condition or, where they are given to bouts of unwarranted irritability.

Depending upon their psychological make-up, and when their condition remains uncomplicated, these patients usually leave the hospital asymptomatic after several days to a week of medical observation. However, I have seen, not uncommonly, cases under this so-called category, who sought treatment at later periods for residual symptoms. The latter, more often than not, reflect psychological rather than neurological symptomatology, with symptoms ranging from poor sleep, fatigability, headache with or without phases of dizziness, disorders of mood, to, inability to function mentally at a satisfactory level. The latter symptom is usually characterized by the sufferer as a tendency to forget and as an inability to concentrate on ordinary work-a-day things.

It is in the latter instance, in this type of trauma, that the medico-legal aspect must be carefully considered and the findings weighed. Complicating issues present themselves clinically both as to diagnostic and prognostic criteria, their role from the standpoint of permanence of symptoms, their bearing upon fixation of causal relation between the accident and symptomatology (subjective and/or objective) and the indications of imitation or exaggeration of symptoms complained of for the purpose of obtaining monetary benefits.

Medical determination respecting the preceding criteria is not easy. The patient's age, state of the vascular system, use of alcohol, presence of lues or other systemic disease, all have a bearing on the final prognosis. In addition, any presenting psychiatric abnormalities should receive, wherever feasible, a special psychological work-up prior to final prognostic determination.

The syndromic picture which may appear after the passing of the acute symptoms following trauma to the head is varied and sometimes considerably troublesome to patients and those who have to live, work, or socialize with them. Moreover, the seriousness of the original blow to the head many times is no yardstick of the measure of sequelae these patients may become subject to. The reason for this discrepancy of proportion will become apparent when we consider the pathophysiological entities and alterations already discussed above.

In patients whom we may assign to this descriptive grouping on the basis of their symptomatologic aftermaths, I have observed subjective and objective evidence of clinical pictures which encompass rather wide territory. Patients may describe such discomforts as vertigo (either positional or rotational), paroxysmal headaches which

give particular discomfort on change of posture (possibly on the basis of vascular stasis predicated upon the cerebral trauma), or may take place after any increased emotional tension. They may show undue fatigue, poverty of concentration, defective memory and photophobia. I have noted ease of excitability in head traumatized patients whose prior personalities were known to have been easy-going, and whose emotional outputs were stable antedating the trauma.

Under this grouping of head injuries therefore, the most likely persistent symptom liability may be headache with or without associated spells of dizziness. Should the latter complaints remain chronic or be made worse on bodily exertion or sudden head movements, meningeal adhesions must necessarily be borne in mind. Clinically however, no definite provable index can be postulated for this premise in the absence of necropsy, inasmuch as EEG tracings do not afford reliable criteria in this class of cases.

In evaluating symptoms and disabilities complained of, the mere length of time that an individual has been away from employment, should not, in and of itself, form a basis for adjudicating him medically as simulating illness. Appropriate work-up and careful medical observation must always remain the criterion upon which to determine whether post-traumatic idleness is predicated upon the patient's psychogenic unconsciously existing symptoms occasioned by his insecurity and conflictful situation or, whether he consciously exaggerates to deceive for the purpose of financial or other gain.

Cerebral contusion sequelae are predicated upon the manner and quality of the skull impact. As in concussion it should again be stated with equal force here that a contused brain need not necessarily be the subject of an unconscious reaction. The location and extent of the lesion is determinative of the character of the aftermaths resulting from contused cerebral structures with or without meningeal or other focal damage.

In this class of closed brain injury, the permanence of the sequel is dependent upon the degree (qualitative and quantitative) of the established brain damage. Inasmuch as no specific visualization of the injured brain is possible in the recovered patient, the unavailability of necropsy makes assessment of sequelae difficult, with determination relegated entirely to the clinical investigation. The latter frequently discloses a long series of symptoms which I have seen to range from: general mental blunting, epileptic seizures, disturbed vision (in brain-stem damage), defective correlation of memory function, headache (localized or diffuse), to dizziness worsened by postural change or any exertion.

From the psychogenic viewpoint, in patients categorizable under this class of post-traumatic disorder, I have repeatedly observed mental slowing for variable periods (the latter extending at times to many months), disturbed sleep, emotional buoyancy coming with depression, sluggishness of reasoning and memory, undue irritability, intolerance to noise, and tension state. The latter sequel when presented, may be ascribable to patients' realization of their lack of integrated mental output. These psychogenic defects, or any of them, are apt to work a more pronounced hardship on those traumatized individuals whose livelihood depends upon concentrated mental function, as compared with others whose employment is of the strictly manual unskilled variety.

In endeavoring to assess the permanence as well as the genuineness of the presented sequelae, medical consideration must include alertness for evidences of motivation. This is of particular importance where tendencies toward stress reaction will reveal in the post traumatized individual a vulnerability and low threshold of tolerance reserve. While predisposition to psychological collapse may be inherent in some patients on the basis of antecedent factors, gleaned from a personal and family history obtained through considered reliable sources, nevertheless no rule of thumb can be postulated which can justify the assertion that a neurotic pre-traumatic inventory necessarily reflects the resultant psychiatric aftermaths. The overall medical determination must be founded upon a totality picture which is to include the quantum of the trauma, pre- and post-traumatic personality make-up, and source of patient's possible motivation. Under the latter I include incidence of financial family-burden

precipitants with inability of the patient to obtain employment as further complicating his situation, or, alterations in the social or domestic sphere which might have developed, or, fears of the possibility of future mental and physical disabilities which may be disturbing. Also, in instances not altogether uncommon during neurological and psychiatric investigation carried out at time of patients' hospitalization, and more particularly in other and later non-institutional settings, I have observed patients after head traumas under this diagnostic category, the character of whose complaints patently essay desire to obtain indemnifying money payments. In the latter instances I have, on occasions, found coloration of the symptomatic picture after objective evidence of craniocerebral disorder has ceased to justify the presented symptoms.

Laceration of the brain is productive of divers aftermaths depending on the extent and location of the cerebral damage. These survivors most commonly complain of headache during their period of convalescence, and more frequently than not, this symptom remains as an indeterminate consequence. I have found that an equally troublesome and lasting symptom is vertigo, which, during convalescence makes unaided walking difficult. During the early part of their convalescence, I have numerously observed in these patients divers exhibitions of retardation of mental activity: thinking may become slowed up, temperaments may exhibit alterations out of all proportion to these patients' pre-traumatic life (as gleaned from informative material obtained from patients' friends, co-workers and immediate families), inability to make responsible decisions may become evident, performance of acts executed without awareness may become conspicuous, coupled with varying gradations of quantitative memory dysfunction (both retro- and antero-grade) depending on the gravity and magnitude of the cerebral damage. While not too frequent, as an incident to this type of brain injury, I have had occasion to observe in patients thus traumatized, instances of the full throes of an acute psychotic state. However, the latter syndromic picture invariably subsides, or only negligible mental residuals may remain as an aftermath, before hospital discharge.[10]

I have had opportunity to study a considerable cross section of patients, both non-military and those returned from the military services of the last 2 wars, as well as the Korean conflict. I have traced their progress and symptoms following periods of months to years after brain injury sustained in civilian life, or as a consequence of cerebral damage by explosive missiles, bayonet penetration, or gun shot wounds. In an appreciable percentage of these civilian and military casualties the aftermath symptoms have included the developing complications of generalized and/or focal convulsive seizures. The variety and severity of these episodes as well as their frequency are predictable upon such variable traumatizing factors as cortical proximity, severity of the injury, cicatrization between dural and brain substance, location of the cerebral lesion, or instances where the etiological factor does not lend itself readily to clinical tracing.

Noticeably also, under this category of encephalopathy, the syndromic picture varies etiologically with the distinctive qualities of traumatized individual, the relationship between the head injury and latent or patent psychotic symptoms at time of trauma (which latter might serve as a precipitant of acute psychotic episode), the pre-existence of disease (degenerative or neoplastic), infectious, toxic or metabolic disease, as well as with the amount and severity of the brain damage sustained traumatically. I have had occasion to observe patients within this grouping with presented symptoms of concentration difficulty; headache complaints which are paroxysmal and made worse on change of posture, upon exertion, or when emotional tension becomes heightened; with unwarranted bouts of anger; with inordinate fatigability; with complaints of vertiginous attacks which these patients will describe as either positional or rotational. I have also seen them in states of deterioration where they reflect evidence of mental confusion; serious and disrupting personality changes; impairment of intellectuality; where they show prolonged confabulatory amnestic psychosis; or evoke a

history of post-traumatic convulsive seizures.[11]

The incidence of abnormal activity patterns as recorded by EEG tracings is all too frequently pathologically inconsistent with the traumatic history and/or the incidence of the physical or psychiatric clinical picture presented. Predicated upon this obmservation therefore, the mere absence of an abnormal, or the presence of an inconsistent EEG record per se, should not lull the neurologist into a prognostic reliance founded upon the EEG tracing, without combined analysis of all other pertinent diagnostic criteria which should in all cases satisfy the weight of prognostic conclusion. "It should be stated at this point that a mere loss of brain substance in a static condition does not produce EEG signs and that therefore the existence of cerebral atrophies and porencephalies should be considered as one of the residual signs of a severe trauma, which still causes abnormalities of cerebral function at the time of the EEG."[12]

Hemorrhage of the brain, from the standpoint of sequelae, depends to a large degree upon the site as well as upon the amount of intracerebral bleeding. The latter need not necessarily be extensive at the time the blow to th head was received. Delayed escape of blood into th brain substance may occur from many causes other than immediate trauma, and, thus delayed, is by no means a rarity as a sequel to head injury. Without detailing here the micropathological alterations of implicated tissues, immediate or delayed bleeding may occur in cerebrally traumatized individuals who suffer from varied physical states and conditions pre-existing the brain injury. Examples not altogether uncommon which I have come upon, and which combine intricately with the current traumatic situation, are circulatory disturbances, diseases of the blood vessels, neoplastic disease, existence of aneurysms or presence of such systemic conditions as lues, nephritis or hypertension. It is such predisposing causes, as may have hitherto produced negligible or no subjective symptoms, that cannot be overlooked or discounted in medically assessing the proximal and latently qualifying factors etiologically accountable for the hemorrhage. This, particularly in instances of late bleeding which may not become evident for from days to weeks after the advent of a head trauma.

The aftermaths of cerebral hemorrhage are divers, and as I have already developed in the preceding categories of brain traumata, are dependent upon many factors which include the site, extent of the area implicated, and incidence of pre-existing disease in the traumatized individual. On the basis of personal observations in this type of injury, I have encountered instances of amnesia, the various gradations of aphasia, facial distortion usually on the basis of 7th Nerve damage, weakness of an extremity, increase of deep reflexes with concomitant loss of superficials, presence of Babinski toe sign and confirmatories, sundry sensory alterations, varied oculomotor disturbances, speech defects, personality changes; and not infrequently traumatic epilepsy or focal seizures also may complicate. Lastly, but rating equal dignity with the foregoing, the subjective complaints of headache of varying severity and subjective localization, coupled with nausea and frequently associated with vomiting are the most commonplace symptoms with which I have been confronted by patients in this traumatic category.[13,14,15]

Discussion

In the determination of valuation of social, personal and economic function of individuals who had been subjected to head traumata, many component factors must be considered—each for its contributing part to the whole presenting picture.

A long range study of a considerable number of post-traumatized patients has served to disclose that recognition of physical or mental incapacity may be predicated upon conscious (*malingering*) or unconsciously (*neurotic*) operating symptoms. Obviously, this problem is most challenging clinically, because criteria must be heterogeneously considered in order to qualitatively allocate the character of the dysfunction complained of. Where it becomes demonstrable that the patient's symptoms were unawarely developed following a head injury, and where they appear to serve to lessen the patient's conscious anxiety and thereby meet his immediate psychic needs, the presenting symptoms

are expressive of an exaggerated state of pathophysiological emotionality not under the patient's normally functioning voluntary perception or control.

As a result of exposure to catastrophic situations such as occasioned by an injury to the head, certain personality types will, not infrequently, exhibit neurotic reaction patterns based upon an overwhelming fear, and reflected as states of anxiety associated with their future problems of many sided readjustments.

While no ironclad correlation is here postulated as between the gravity of the initial trauma and the severity of the morbid consequences thereof, I have found that considerable correlation does exist between the presence of such complicating situational factors as actions at law, contemplated litigation predicated upon the alleged liability of another, unliquidated compensation payments or awards, concomitant traumata to other bodily parts, and the perseverance and distinctive character of existing neurotic consequences. The latter, together with the patient's conjectured cerebral effects, are not infrequently employed by the neurotic personality (without awareness) as an escape from fear, panic, or accountability for past deficiencies, and as a protective covering for lethargic response to social, economic and personal readjustment.

From the writer's observations of disabilities following cerebral traumata I have found it very helpful prognostically, to evaluate as far as possible the individual's pretraumatic psychic status, to take into account the patient's adaptability, initiative, aspiraions, intentions, and their concrete results before the injury, and to recognize any current alteration in the totality of his surrounding circumstances and/or expectations since the injury.

Frequently a patient's desire to obtain money payments as indemnification appears to color the symptomatic picture—and not infrequently the issue operates to clinically complicate it—when objective clinical evidence of craniocerebral disorder ceases to justify averred post traumatic residual symptoms.

On the other hand, where the presenting syndrome picture is demonstrably predicated upon psychic or psychophysiologic factors unawarely evinced, the neurosis may be founded upon the patient's presently-acquired anxieties, upon threats to his personal and/or family security, loss of employment, domestic or legal difficulties arising from his accident, financial problems, social dislocations, and the presence of other diseases or of concomitant injuries which the patient imputes to his head trauma. I have found that the manner in which the trauma was sustained—compensable accidents (causality of which may be chargeable to another) in contrast to accidental falls in the home, injuries during engagement in sports, hurts in altercations or in hold-ups—not infrequently provides a significant connection between the question of compensation for personal damage, its effect upon the patient's disablement, as well as upon the issue of his behavior pattern.

However, the clinician's determinative task becomes most exacting in situations where a patient does not demonstrate signs of adequate expectant recovery from a specific cerebral trauma, though a variable and obviously extended period of time may have elapsed since the injury. The latter may be followed by persistent anxiety and dispiritedness where the patient believes them to be solely the effect of the head trauma. This syndrom may have insinuated itself as a newly acquired psychophysiologic nervous system reaction, or the trauma may awaken latent predisposition to psychophysiologic disorder, when economic insecurity with inability to obtain work, or when alterations in the social sphere, or disruptions in the domestic sphere, have developed.

A long-range study of considerable numbers of post-traumatized patients has served to disclose to the writer that where psychophysiologic posttraumatic syndrome exists without clinically demonstrable dysfunction, it is the rule rather than the exception that the post-traumatized individual who is left with no residuals of organicity is basically a neurotic personality who had been experiencing difficulties in dealing with situations of lesser gravity or importance, and who now endeavors to utilize his traumatic experience to resolve his latent incapacities and unsolved problems.

Although malingering is held by many to be a neurosis, and mindful of the dignity accorded it in a not inconsiderable amount of psychiatric literature, the writer is of the opinion that this type of "neurosis" (without here posing its psychological pros and cons) merits no premium to be placed thereon. Accordingly, I feel that once found to exist per se, the individual warrants no medical evaluation within the sphere of psychological appraisal conducive to compensation or indemnification therefor, whether by award, litigation, settlement, or otherwise.

Malingering of symptoms ascribable to the nervous system imputed by the patient as a result of head trauma is a medically challenging situation, the recognition of which requires meticulous objective attention to the dysfunctions displayed and the disabilities complained of—procedural detailing of examination for which, will not be entered upon in this study. Inasmuch as this controversial subject frequently presents many contested syndromic factors, their recognition plays a further important role from the determinative angle in medico-legal situations bound up with the question of compensability, liability, indemnification, etc. It should be noted in dealing with this situation and where its consideration is contemplated from the etiological standpoint, that it is well to keep in mind that not all head traumata may be invariably attended by histopathologic tissue alterations, although an ephemeral cerebrophysiologic condition may be in evidence at the onset of skull impact.

Conclusion

In keeping with the scope of the subject matter and its discriminative application to the many sided aftermaths which have been respectively dealt with as occurring after particularized head injuries, reference to specific cerebral localizations and related treatment had not been included in this treatise.

Traumata to the skull and/or brain may precipitate neurotic changes in certain instances or may extend into lasting pathological alterations with implication of ganglion cells, nerve fibers, glia, blood vessels, and meninges. Laceration and hemorrhage with areas of cerebral softening may occur and varying gradations of encephalopathies and secondary degenerative changes may similarly complicate the picture.

The writer's observations have been presented, discussion of specific head injuries and their sequelae has been directed toward descriptive allocation and assessment of their neurologic and psychophysiologic patent and latent effects within specific categories, and the divers physiogenic and psychogenic dysfunctions as aftermaths flowing therefrom have been evaluated.

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Dormison as an Adjuvant in Electrocoma Therapy

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Fractures and the fear of shock are recognized drawbacks to electrocoma therapy (ECT) that often prevent its use. Fractures represent an unwelcome and handicapping complication. Some patients who would benefit from electrocoma therapy refuse it because of fear. Others discontinue psychotherapy when this treatment is suggested. The fear of shock has such great psychological effects that it may render electrocoma therapy unacceptable to the patient, or to relatives who usually authorize its application, or may even nullify its benefits.

We have attempted to find methods of overcoming the shock phobia or fear reaction to electrocoma therapy. In at least 50 per cent of instances when we have used it despite all drawbacks, doctor-patient relationships have deteriorated for the physician administering it and for other therapists. As a consequence, psychotherapy and rehabilitation have become more difficult.

Among methods heretofore employed to exorcise this fear, the most common is reassurance. This is sometimes wrongly carried to the point of making the patient believe he will not feel the current. In our experience, reassurance succeeds only in patients "in contact" and with appreciable insight or in withdrawn catatonic and depressed patients. Others are seldom convinced and frequently respond with antagonism, secondary reaction formation, and stubborn passive resistance.

Brody[1] has used sodium thiopental narcosis to overcome the fear reaction prior to applying the current. Kalinowsky and Hoch[2] found no advantage in premedication and many disadvantages such as increased risk of bulbar respiratory arrest, hypersecretion of mucus, and memory of slowly inducted current and semiconscious panic while waiting for it. They wisely state that it is a mistake to promise a patient medication to make him unaware of treatment. Other therapists have used various barbiturates. In susceptible patients, barbiturates may cause secondary reactions that add unnecessary complications to the procedure. Furthermore, a number of patients are dizzy and giddy on awakening and require watching for a prolonged period—anathema in institutional practice. The use of ethylene or chloral hydrate is even less propitious. Hypnosis has been used, notably by Maholick and Warkentin[3] and Sagredo,[4] but this procedure is practical for only a few workers.

New Hypnotic

Dormison, a new hypnotic of low toxicity which is non-habit-forming, and relatively innocuous,[5-14] was tried by us shortly after its release. We administered it with the idea of inducing deep hypnosis to insure that patients would be unaware of the preliminaries to electrocoma therapy. We expected the drug to cause no respiratory depression, cardiac irregularities, hangover, or neurological changes since other authors had reported their absence.[5,10-13,15]

A group of 24 psychotic patients of both sexes received Dormison. Among them were 3 with manic depressive psychoses and 21 with schizophrenia. Eight of the patients had previously had electrocoma therapy; 3 of them had required heavy barbiturate sedation to overcome resistance to the treatment. Of the 16 untreated patients, only 3 were not fearful (2 catatonic and 1 depressed patient); 5 were extremely resistive to ECT.

The optimum dosage of Dormison was first determined by its use during preparations simulating those before electrocoma therapy. Breakfast was withheld from all patients or limited to a glass of fruit juice. They were put to bed in pajamas at 9:00 A.M. Pulse rates and blood pressures were taken before and at 15 minute intervals after the administration of Dormison. Blood counts and urinalyses were performed periodically. Dormison was administered when the patients had adjusted to being put to bed. With a

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dose of 500 mg., only 2 patients with mild schizophrenia went to sleep; with 1.0 Gm., 3 more slept. A dose of 5.0 Gm. induced sleep in 6 additional patients, but 4 awoke at the slightest touch. Laboratory examinations and clinical observations revealed no untoward reactions to the drug. The large dosage required to achieve sleep in these psychotic patients was considered too great a drawback to the use of this method preceding therapy. Our results were not surprising inasmuch as it had previously been found that larger than average doses of Dormison were required to induce sleep in acutely agitated or disturbed patients.[11]

Comparison of Dormison with Secobarbital

For comparison, secobarbital was tried in the same 24 patients. Three, six and four slept with gr. 1½, 3, and 6, respectively, administered orally. Nine patients slept with a dose of gr. 7½ but could be aroused. Two patients in a state of excitement failed to sleep but were sedated with gr. 7½ amobarbital sodium intramuscularly. Table 1 shows the numbers of patients asleep with the various doses of Dormison and secobarbital.

TABLE 1.

MINIMUM DOSES OF DORMISON AND SECOBARBITAL REQUIRED FOR HYPNOSIS IN PSYCHOTIC PATIENTS

Dormison		Secobarbital	
Minimum Dose	*No. Patients*	*Minimum Dose*	*No. Patients*
500 mg.	2	1½ gr.	3
1.0 Gm.	3	3 gr.	6
3.0 Gm.	1	6 gr.	4
4.0 Gm.	2	7½ gr.	9
5.0 Gm.	6		
Total patients asleep among 24	14		22

Careful observation of patients showed that sleep induced by Dormison lasted an average of two hours. All patients could be aroused without difficulty. Drowsiness, torpor, incoordination, nausea, and vertigo were minimal or absent following the use of Dormison. Two patients who received the extraordinarily large dose of 7 Gm. complained of pyrosis.

Patients receiving secobarbital slept an average of three and three-fourths hours. Some could be aroused with difficulty before the end of that time. Eighteen patients were drowsy for at least half an hour after awakening. Two patients showed torpor and 5 incoordination. Three patients vomited, 5 had nausea, and 1 vertigo.

Table 2 contrasts the side effects encountered with the two drugs. Dormison was definitely superior in regard to lack of side effects.

TABLE 2.

SIDE EFFECTS OF DORMISON AND SECOBARBITAL

Drug	Dormison	Secobarbital
Average Time Asleep	*2 hours*	*3¾ hours*
COMPLICATIONS:		
Drowsiness	3	18
Torpor	–	2
Incoordination	–	4
Nausea	–	5
Vomiting	–	3
Vertigo	–	1
Total Complaints Among 24 Patients	3	34

Sedation with Dormison

While the preliminary investigation revealed that psychotic patients often did not sleep even with high doses of Dormison, it was noticed that 3 of the 5 patients previously afraid of electrocoma therapy became calm and started to accept the idea of receiving treatment. As the dose-determining test was not followed by actual therapy, this behavior may have resulted from its absence although the change was noted from the first day. Treatment was initiated for these patients and they accepted it then and subsequently with little or no apprehension. The 2 patients who showed no change were in a state of manic and paranoid excitement that rendered them hostile to the environment.

Questioning of patients in the experimental group who were "in contact" disclosed a noticeable decrease in anxiety and apprehension. Examination of the behavior of the remainder also showed decreased resistance to the preparations for ECT and the treatment itself. None of the patients manifested depression, mental dullness, euphoria, exhilaration, or hilarity. Most of them summarized their subjective feelings with the statement, "I feel all right." This picture presented a contrast to that of patients unable to sleep after secobarbital. Most of the latter were dazed, restless, and apprehensive and required frequent reassurance.

The absence of abnormal clinical and lab-

oratory findings following the use of Dormison has already been noted.

In contrast to barbiturates, Dormison caused no appreciable changes in pulse and respiration rates. Cardiovascular functions did not vary. The convulsive threshold following its administration was not noticeably changed.[16] Dormison did not influence nor alter the muscular convulsion following electrocoma therapy.

Dormison for Sedation in Electrocoma Therapy

The decrease in anxiety and apprehension effected by Dormison was considered more valuable than inducement of sleep preceding electrocoma therapy. Patients remained awake and cooperated better, closer rapport could be established, and the drug appeared to be free from undesirable side effects. The study was, therefore, continued following this first experience. It is interesting to note that Malone[13] found Dormison a satisfactory sedative in restless, agitated, and mentally deficient children. Tukel and Tukel[17] employed its sedative effects to calm children prior to electroencephalography. The drug did not affect the basic electrical activity of the brain.

Six psychoneurotic (anxiety reactions 3, conversion reaction 1, phobic reactions 2) and 26 psychotic patients (organic psychoses 2, manic reactions 3, depression 4, schizophrenia 17) were given electrocoma therapy at various times. When contact was possible, all patients were given assurance that the treatments would not be harmful and in most cases the electric current would not be felt or, if felt, it would be for a brief moment. All of the psychoneurotic patients were apprehensive. One was fearful, and one required thirty minutes' persuasion before accepting the first treatment. Five psychotic patients (manic reactions 2, depression 1, paranoid schizophrenia 2) were extremely resistive, the remainder submissive.

Before the second treatment, 5 of the 6 psychoneurotic patients were more apprehensive or fearful than previously. Four requested sedation or the inducement of sleep before the treatment. One refused ECT. The resistive patients were again reassured and received 3 to 5 Gm. Dormison before the treatment. Dormison was explained as a medication to relax the patient and minimize his apprehension. As a result of our preliminary test, it was thought that patients as tense as these would not go to sleep on these doses, and they did not. However, the 4 patients requesting sedation became calmer half an hour after Dormison administration. The one who had refused therapy requested an additional dose (500 mg. extra was given). All submitted readily to treatment. Questioning of these patients the day after ECT showed that the 5 psychoneurotic patients still did not like ECT but were no longer afraid or accepted it. The one remaining, who had a phobic reaction, expressed his rejection of the treatment by the rationalization that, "It caused too many physical disturbances and might harm me."

The 5 resistive psychotic patients remained so and 2 would not lie down for the second treatment. Following 3 to 5 Gm. Dormison, all 5 showed greatly decreased resistance and readily submitted to treatment within three-quarters of an hour. Subsequent questioning showed these patients to be less antagonistic to ECT. They did not object to the subsequent treatment.

These findings confirmed the preliminary observation that Dormion possesses anxiety-allaying properties. Dormison has since been administered to 2 additional psychoneurotic and 20 psychotic patients who were apprehensive, fearful, or resistive to treatment. Nineteen responded well to Dormison given one-half hour before therapy. The remaining 3 were still apprehensive but submitted to treatment without rebelliousness. All except 4 patients remained awake after the administration of Dormison.

Discussion

Paralleling this study, the reactions of other patients receiving electrocoma therapy were observed. Apprehensive or fearful patients treated without sedation remained antagonistic. Some patients required narcosis so that treatment could be given.

In all patients except those with extreme excitement requiring barbiturates intravenously for hypnosis, we found that Dormison allayed to a great extent the fearful reactions aroused by impending electrocoma therapy. Patients remained awake, were

rendered agreeable, and cooperated during their treatment. Better psychological rapport, a factor of utmost value in post- treatment rehabilitation and reorientation, could be established. No undesirable complications were found with Dormison. Patients had no hangover and were immediately able to resume their daily routine. This suggests application of the method in ambulatory outpatients.

The minimal effective dose for sedation was found to be 40 mg./lb. If an adequate response was not obtained in half an hour, a supplementary dose of 20 mg./lb. could be given and electrocoma therapy administered one-half hour later. Ten per cent of patients proved refractory to Dormison. This percentage has recently been reduced to 4 per cent by combining the minimum effective dose of Dormison (40 mg./lb.) with Mephenesin (1.0 Gm./50 lb.) in elixir form, both given orally one-half hour before treatment. Mephenesin is nonhypnotic but a powerful relaxant and appears to potentiate the action of Dormison.

Summary and Conclusions

The administration of Dormison to 46 psychotic and 8 psychoneurotic patients one-half hour prior to electrocoma therapy eliminated apprehension, fear, and resistance to therapy. Better psychological rapport could be established and at the same time the traumatic experience of electrocoma therapy itself was minimized. Neither undesirable side effects, such as encountered with barbiturate sedation preceding electrocoma therapy, nor complications were found with Dormison. The drug is considered safe even in massive doses (7 Gm.) for short-term use, *i.e.*, every other day for four to eight weeks. In severe cases of apprehension complicated by excitement and rebelliousness, combination of Dormison with Mephenesin is recommended.

Authors' Note: Dormison was furnished by M. William Amster, M.D., Schering Corporation, Bloomfield, N. J.

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Results of Lobotomy on Seventeen State Hospital Patients

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This paper is a report on 17 patients of the Topeka State Hospital who received pre-frontal lobotomy. The principal goal has been to assess the results and compare them with those reported elsewhere, to review the criteria used for selection of cases, and to make suggestions about future lobotomy evaluation.

There are 17 patients of this hospital on whom lobotomy has been done and who have now been followed for periods varying from 14 to 43 months. These patients ranged in age from 27 to 70 at the time lobotomy was done and there were 15 females and two males. There were two cases of tabes dorsalis with intractable pain, one case of senile depression, and fourteen cases of schizophrenia. The duration of illness before lobotomy varied from four and one half years to 23 years. All of the patients were considered to have had adequate courses of all the known treatments available including literally all forms of anti-luetic treatment for the tabetics, EST for the depressed patient, and insulin, electric, and metrazol shock for the schizophrenic patients. Adequate psychological testing was done before and after lobotomy in six cases. The follow-up psychologicals come between one and two years after surgery. Actually psychological tests were attempted on more patients but often the extremely disturbed state of the patients prevented adequate testing before surgery. There was one case in which the patient was actually testable before surgery but uncooperative afterwards. The clinical status before surgery varied somewhat. Crises of abdominal and leg pains requiring opiates for even minimal relief and apparently great physical discomfort characterized the two tabetic patients. Mental pain, established chronicity, and the failure of less drastic therapies seem to have characterized the schizophrenic group as well as the one senile depression. The patients were considered in mental pain when combative, agitated, destructive, hyper-active, sleepless and otherwise bothered by delusional and hallucinatory experiences. These characteristics comprised the principal criteria for selection of cases. All of the patients received a pre-frontal lobotomy using the Lyerly-Poppen technique. The first five lobotomies were done at local city hospitals and the other twelve were done on the surgical ward of the Topeka State Hospital. Following lobotomy there was close follow-up on both men and one of the women in various wards of the hospital. The other 14 were subjected to an active retraining program on one ward of the Women's Section. This program was curtailed in the summer of 1951 due to the loss of some of the personnel actively engaged in what was called the lobotomy total push program. The program was further curtailed in the fall of 1951, due to the extreme shortage of aides and other auxillary personnel. By November 1951, the only remaining group activity for the post lobotomy patients was the program conducted by volunteer workers. These volunteers received constant encouragement from the hospital staff and made a tremendous contribution when many other activities were at a stand-still. With the help of our own adjunctive therapy and aide staff, they promoted group projects such as gardening, sewing, and social parties. On one occasion a window shopping trip down town was carried out. In December 1951 it was decided that, except for the volunteer post lobotomy group, patients would be treated on an individual basis working them into activities and projects that were available to all patients. All accessible patients received psychological tests and treatment plans were made to fit the needs of each patient. This more or less individual approach is still in operation.

Results

Of the total series of 17, four patients, one tabetic and three schizophrenics are at home. This is an over-all at home rate of 23.5 per cent and 21.4 per cent for the schizophrenic patients. The tabetic patient although withdrawn, apathetic, and at times bothered by abdominal pains, is still able to live at home with his mother. He does no work.

Of the three schizophrenic patients at home, one resides with her parents on a farm doing useful housework. She is expected to go out to work soon. Our chief psychologist followed this patient in individual psychotherapy from December 1951 to the present time. The results were truly astounding. Another of the three now at home lives with her husband and takes fair care of her home. The third is living with her parents, sews, cooks, washes, cleans and enjoys trips to town. It is recognized that all of these cases are leading dependent existences and will probably continue to do so.

Of the remaining 13 patients, five have made moderate improvement in that they no longer are in pain, they are cooperative, are not destructive or combative, and are making a fairly satisfactory hospital adjustment. Two of these patients have been unsuccessfully tried on visits to their families. One of the five had received only one half of a one sided lobotomy due to extensive hemorrhage at the time of surgery. Her result, however, has been fairly good. This makes a total of nine patients or 53 per cent of the total series who are either at home or making fairly good hospital adjustments.

Of the remaining eight patients, four have made mild improvement in that their response to hallucinations and delusions is not as forceful and they are more cooperative. This comprises 23.5 per cent of total cases.

Two patients, or 11.7 per cent are not improved. Even though their pain is gone, other complications cancel out the gains. One, a schizophrenic, showed increase in lying, drinking, and social irresponsibility. The other, a tabetic, developed chronic granulating ulcers on the buttocks and has remained a chronic medical problem even though her physical pain is minimal.

Two patients, or 11.7 per cent died. One died four months after surgery of infections or toxic hepatitis. The other died sixteen months after lobotomy of nephrotic syndrome. This last patient was a known diabetic of many years' duration.

None of the surviving patients were made worse as far as their pain and discomfort is concerned. None of the patients could in any sense be considered cured.

Discussion and Conclusions

None of the lobotomized patients were seen by the writer pre-operatively and consequently comparisons could only be made between the pre-lobotomy condition described on the charts and what the writer and others could observe post-operatively. The defects in this method of evaluation are recognized. Even after allowance for the zeal of the lobotomy advocate in presenting the pre-lobotomy picture the results are satisfactory.

Among the schizophrenic patients, the younger patients and those who had been sick the shortest time achieved the most improvement from lobotomy. This tends to corroborate the work and reports of others.

Also the more tense, disturbed, combative, and bothered or "tortured" by hallucinations and delusions the patients were before lobotomy the better the result of surgery. The patients who were not so bothered and tense did not seem to gain as much. This confirms other reports and tends to support our criteria for selection.

The percentage of patients (53 per cent) who are either now at home or making a moderately improved hospital adjustment compares favorably with other reports although the actual percentage (23.5 per cent) who are now at home is slightly less than other series. Probably more patients will leave the hospital as time passes as there seems to be improvement still taking place in several of the patients.

An interesting observation has been that in all four of the cases who are now out of the hospital and living at home the relatives in each case are accepting the patient and his or her dependence. In the case of the two schizophrenic post-lobotomees who were paroled but returned to the hospital the rel-

atives were not too coperative. The family of one patient repeatedly complained about patient's slowness and would not allow for her dullness and lack of interest. The husband of the other patient, himself, a very dependent man, leaned heavily on the patient and loaded her with responsibility in spite of advice to the contrary. These results support previous observation that accepting relatives are necessary in order for adequate adjustment at home to take place.

Changing personnel will undoubtedly continue to be a problem both with the remaining post-lobotomees and future ones. It would, therefore, seem advisable to set up definite standards for clinical and psychological evaluation so as to make accurate comparisons between pre- and post-lobotomy status.

In addition more attention should be paid to the relatives of pre- and post-lobotomy patients with a view toward early utilization of accepting relatives and possibly work with others to reinforce what capacities for acceptance are present. The attitude of patients' relatives should be taken into consideration at the time the patient is being selected for lobotomy.

Summary

1. Seventeen patients have been observed for periods varying from 14 months to 43 months following lobotomy. Fourteen of these were schizophrenic, two tabetic, and one depressed.

2. Four patients are at home and five more are making fairly good hospital adjustments. Four others are slightly improved, two unimproved, and two died.

3. The younger the patient, the shorter the duration of illness, the more disturbed the patient, and the more "tortured" he is by delusions and hallucinations the better the lobotomy results.

4. In the case of the four patients now at home an important factor seems to be the accepting attitude of relatives.

5. Definite standards of clinical and psychological measurement should be set up in order to make evaluations of results more accurate.

6. Attention should be paid to relatives with a view to using the accepting ones and evaluating their attitude toward the patient at the time the patient is considered for lobotomy.

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Carbon Dioxide Convulsive Treatment

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I intend to discuss a technique in the treatment of the neurotically depressed patient who does not react quickly to the orthodox carbon dioxide inhalation therapy. I utilize the carbon dioxide inhalation to the point of convulsive reaction in these cases.

In order to do this a number of factors had to be worked out, including an exact understanding of the end point of nitrous oxide inhalation given before the regular carbon dioxide treatment, the end point of the regular carbon dioxide treatment, and the end point of the convulsive therapy.

Meduna reports his use of nitrous oxide for a period of 30, 40, or 60 seconds, having determined experimentally with each patient the length of time required to render the patient unconscious. He speaks to the patient until replies from behind the mask cease. He estimates the amount of time required to this point as the end point for the nitrous oxide.

I usually go a little further than the end point of non-response and wait until other signs appear. One is the jactitation sign which occurs with cerebral stimulation and can be observed by watching the hands of the patient; the hands begin to tremble coarsely and the body may do the same. Another sign is the complete relaxation which comes slightly before jactitation. This can be observed in two different ways: when the patient's arm can be lifted up like a mass of putty, then falls limply; the second is the relaxation of vocal cords, soft palate and pharyngeal muscles. If this sign appears one can practically always hear snoring. The easiest clinical sign to look for is the snoring.

The ordinary signs of anesthesia such as inward and downward deviation of the eyes and pupillary reactions cannot be seen as the mask usually covers the face.

In giving nitrous oxide inhalation before the carbon dioxide we had little trouble in knowing when unconsciousness was first achieved. Without the nitrous oxide preceding the carbon dioxide the therapist can count the breaths the patient is taking in a loud voice and when the patient seems to be breathing automatically he removes the mask. When the patient becomes fully conscious he can usually tell the last number he heard. At the next treatment he is given two or three breaths more than the last number, thus making sure he has received sufficient gas to render him unconscious.

With the nitrous oxide given first the patient is unconscious and cannot hear the count. Therefore, we must have objective signs to indicate that the patient is unconscious. There are two major signs and one minor sign that I have found of value. First there is a mild quivering tremor of the finger tips, not the coarse tremor, taking place in jactitation. The second major sign is a marked rigidity of the limbs, so that when attempt is made to lift the upper extremities they come up with great difficulty and do not bend at the joints. The minor sign is the mechanical breathing. At the time the change is made from nitrous oxide to the carbon dioxide mixture the patient takes about 3 or 4 breaths of carbon dioxide, becomes flushed and then has an apnea lasting from 2 to 6 seconds; then he gradually starts breathing deeply and mechanically.

The convulsive approach is experimental and I have used it only in nine cases, four of whom have showed clinical recovery. They were severely depressed patients who received carbon dioxide inhalation therapy preceded by nitrous oxide to make it possible for them to take the treatment. The carbon dioxide was given to the point of unconsciousness only at the beginning. After they had from 16 to 34 treatments without any marked improvement I decided to use the convulsive approach and had four recoveries which came after three to four treatments. However, treatment was continuing for a while in each case in order to make sure that the results were permanent. The method

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was to continue the carbon dioxide after the patient reached the level of unconsciousness. From the slight quivering of the fingers there was gross quivering and then some clonic reactions. I found frequently that the patient did not go on to any convulsive reaction aside from the clonic movements. I then tried giving an additional 10 breaths and found that the patient would usually go into a strong clonic reaction lasting from 30 to 45 seconds, followed by a tonic convulsive reaction, and then by a clonic reaction. The tonic phase would last about three to five seconds and would be accompanied by apnea. Clonic reaction at the end would last anywhere from 5 to 30 seconds.

The convulsive reaction had certain peculiarities. It was not anywhere near as severe as in metrazol or electro-convulsive shock. The patient frequently achieved a degree of consciousness during the second clonic phase and might start looking around and occasionally might be frightened. However, I would talk to the patient, reassuring him with a hand on the shoulder to establish contact and rapport which usually calmed him.

The number of breaths required to bring out the convulsive reaction varied. For instance, two years ago while administering carbon dioxide I inadvertently got a convulsive reaction when the patient took three breaths. The E.E.G. of this patient had never shown any abnormality, but I do suspect that this patient had an exceptionally low convulsive threshold and possibly was a latent epileptic. The amount of carbon dioxide required for convulsive reaction is usually 48 inhalations or more. The average dose required to get a convulsive reaction in the four patients who recovered was 62. This seems a great deal, but in some patients it is possible to go much higher. I have gone up to as high as 98 inhalations before getting convulsive reactions.

Now, as to the manner of handling the patient during a convulsion. Since it is not a severe convulsion it is not necessary to do much. The patient should be left pretty much alone. He is placed on his back, a cushion under his head. One person can handle the situation if the table is against the wall. Care must be exercised that he does not fall off the treatment table or bite his tongue. Special padding in the lumbar curve is not needed. The patient need not be restrained in any other way. I have not had any fractures.

Another group resistant to carbon dioxide are the obsessive-compulsive cases. However, I have found these reactive to the CO_2-O_2 treatment under certain conditions. As an experiment I had a group of three that I treated. In these cases, all adults, the patients had suffered from the obsessive-compulsive reaction from one to three years. Two were scheduled for lobotomy and were sent to us as a last resort before lobotomy. The attitude was, "It can't do any harm. We are going to dissect their brain anyway, so let's see what the carbon dioxide can do." I informed the family and referring psychiatrists that we could not expect too much because we had not gotten very favorable results in obsessive-compulsive cases that had received 50 or more treatments. If there was improvement we would have to go into a field that had not yet been explored thoroughly; that treatments at the rate of three a week would have to be given for at least a year. I also explained that it would require the cooperation of the whole family to get the patient to continue the treatments.

These patients were treated for approximately a year and the results in all three cases were excellent. One patient started therapy in March 1951, and treatments were terminated in April 1952. Another was treated from June 1951, and treatments were terminated May 1952. The third was treated since April 8, 1951. Two have recovered completely as far as necessity for treatments is concerned. The third is still under treatment. Two have occasional periods of mild anxiety as a result of obsessive feelings, but they can control them; the same is true of the third case, but he has requested continuation of treatment because of the fine results. We hope eventually to eliminate even the transitory anxiety spells. All these patients can work regularly, they enjoy living, and are capable of taking their place in all social activities normal for their group. They are happy they didn't have to go through a lobotomy.

It is interesting that all three patients had

psychoanalytic treatment in the hands of excellent analysts in the city of Chicago for a long period; one for eight years, without improvement. This patient was referred by this analyst.

I would like to mention also the use of carbon dioxide in epilepsy. I have tried it on a number of cases, but have not been able to follow through for one reason or another. It seems, perhaps, anomalous that an anesthetic should be used to prevent a grand mal seizure. It isn't when one considers that some clinics, especially in England, are using electric shock for prophylactic control of convulsive seizures. The idea, originated in English clinics, was to give epileptic patients who were resistant to pharmacological treatment an electric shock treatment about once a month, or once every two weeks, to prevent occurrence of convulsive seizures in an unsafe or dangerous place. This is done by exploding the accumulated cerebral electric charge in a proper and safe clinical set-up.

One paper has been published on the use of carbon dioxide in epilepsy.[1] The authors reported ten cases of epilepsy which had been treated pharmacologically with the usual medicaments without improvement. They were the dregs of a group that had been treated. The other cases had responded and these had not, but they did benefit from the additional CO_2 therapy. I tried carbon dioxide on several patients over a period of three months. Two of them I was able to treat consistently and they showed marked improvement. One patient had been having two or three seizures a week and these were cut down to once a month.

I did not go to the convulsive stage in treating epilepsy with the carbon dioxide, only to the stage of unconsciousness. Perhaps it would be possible for some work to be done whereby the patient takes some carbon dioxide inhalation every morning, say to the point of dizziness, and this might be sufficient to cause a discharge for that day of the accumulated electric charge in the brain so there is no build-up with resultant grand mal reaction.

1. Baudouin, A., Remond, A., and DeLarue, R.: Therapeutic Effects of Inhalation of Oxygen-Carbon Dioxide," *Bull. Acad. Nat. Med.*, Paris, Vol. 131, p. 483-487.

In treating the obsessive-compulsive cases I used the routine carbon dioxide therapy to the point of unconsciousness, not to convulsiveness. These cases were what is classified obsessive-compulsive neurosis in the American literature. However, they all started in adulthood and are probably slightly different from the group mentioned by Meduna that started in adolescence. However, I have had some disappointing experiences in giving 40 or 50 treatments to obsessive-compulsive cases and seeing absolutely no result whatsoever. It was for that reason I undertook the treatment of these patients with the understanding that they would go along for at least a year. The reason the patients stuck to the treatment was primarily a push from the family. They were usually started off with five treatments a week. I never saw anything in the way of improvement in them with less than 100 treatments. My impression is that to get a change in obsessive-compulsive cases it is necessary to give the patient at least a year of treatment.

In a paper I read in May 1951 at the A.P.A. meeting I mentioned that carbon dioxide had an effect not unlike a chemical lobotomy. I still think so. I do not think each individual treatment causes much change, but there is a cumulative result. It does not seem to diminish intelligence or damage the memory. In that way it is superior to the surgical lobotomy. Undoubtedly there are obsessive-compulsive cases that will be refractory to prolonged carbon dioxide treatment. I have been fortunate in the small number I have treated.

Discussion

Dr. Ludin: What Dr. Jackman spoke of in his treatment of compulsive-obsessives is very interesting. I have treated several compulsive-obsessives who have gone through courses of shock treatment without any cessation of their symptoms with E. C. T. or with CO_2.

I have been able to give these people some amelioration of tension, but not of other symptoms; however, one woman whom I treated for 70 consecutive days with CO_2 followed by two shock treatments at the end of that period began to work on returning home and is still working some six months later. It was the first time she was able to work in three years.

Dr. Joseph C. Borrus: Did you carry the treatment of the obsessive-compulsive patients to the convulsive level?

Dr. Jackman: No, just the routine unconsciousness.

Dr. L. Meduna: I would like to call attention to the different classes of obsessive-compulsive neuroses, because I am sure there are various states included under this name in the same class.

There is a type of obsessive-compulsive neurosis that is called in Europe, "anancastic" neurosis. The adjective anancastic is a derivative of the Greek work "Anake" which means Fate, or more accurately, the Goddess of Fate. The name anancastic neurosis implies that the neurosis is controlled by irresistible and foreordained forces. This anancastic neurosis classically begins around the age of sexual maturity and proceeds irresistibly, relentlessly enveloping in greater and greater circles the patient's activities until he dies.

Now, apparently this was the group I diagnosed obsessive-compulsive neurosis and treated with CO_2 but there was nothing approaching improvement in this group.

There is, however, another group of patients with obsessive-compulsive reactions in which the relation of this *reactive* obsessive neurosis to the classical anancastic neurosis is not unlike the relation of a schizophreniform reaction to schizophrenia. The former is either self-limiting or easily curable, the latter has a bad prognosis.

In the particular group of reactive neurosis of obsessive-compulsive type I was able to produce some improvement, but I do not consider these patients as belonging to the group of anancastic neurosis. I believe this minute distinction within the group of obsessive-compulsive neuroses is the cause of the difference of opinions regarding the prognosis in this disease group.

Dr. Wilbert M. Gansloser: We have been using this technique since 1948 and we have had no results with obsessive-compulsive neuroses although we have taken them to the convulsive level for 50 to 100 treatments. Interestingly enough, those patients who had prior psychoanalysis seemed to respond more quickly with less intense carbon dioxide than those without.

Dr. Jackman: I do believe the cases I treated were classical obsessive-compulsive neuroses. I have had poor experience with this group if I gave only 50 to 70 treatments. For that reason I undertook the treatment of a selected group of obsessive-compulsive neurotics with the plan that the patient would receive treatment for a whole year.

The patient usually started off with five treatments a week for about four months, then the number of treatments was decreased to three treatments a week. The number of treatments reached to the hundreds.

The cases I have treated in this way had received every possible treatment before and they were selected for frontal lobotomy, but the patients and their families felt prompted to have them undergo the experimentation with carbon dioxide; the patients were usually brought in by their families. I did not see any improvement in these cases in less than one hundred treatments.

If you are willing to undertake the experiments with the obsessive-compulsive group, you would have to give many hundreds of treatments and it would certainly take a year or more to see any results.

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. 510435

Presentation

A 75-year-old white widow, quite obese, was admitted to the hospital for the eighth time on April 10, 1951 and died April 24, 1951.

In January 1940 the patient had been admitted for a Pott's fracture of the right ankle which reduced easily and healed well although she cried and screamed for the first 24 hours in the hospital.

In August 1941 at the age of 65 she was admitted again with the complaint that for 2½ weeks she had been aware of gradually increasing weakness of the upper extremities, numbness over the radial aspect of the left forearm, hand, and of the third and fourth fingers. Physical examination revealed great weakness of all movements of the upper extremities excluding those of the shoulder girdle, mild atrophy of the thenar and hypothenar eminences and flabbiness of the other muscles of the upper extremities bilaterally. There was a questionable slight weakness of the lower extremities. The deep reflexes were present in the upper extrmities and absent in the lowers. Blood pressure was 140/90 and heart rate 80. Thorough analyses of the cerebrospinal fluid done on this admission and subsequently revealed no abnormality; they included studies for subarachnoid block. A blood Kahn was negative. Peripheral blood was as follows and remained essentially the same for the rest of her life: RBC 4,430,000, hemoglobin 12.4 Gm., WBC 4,500; differential: polymorphonuclear cells 48, lymphocytes 46, eosinophils 6. Urinalysis was not remarkable. Roentgen examination of the cervical spine on this and subsequent hospital studies showed hypertrophic arthritis, the space between the 6th and 7th cervical bodies being narrowed. The patient was given vitamin preparations and liver extract; she improved slightly and was discharged 4 days after admission.

There was no family history of muscle disorder save the diagnosis of tabes dorsalis made in the case of the patient's father who had died in middle age of carcinoma of the prostate gland and multiple pulmonary infarction. The patient had had 3 children, 2 male and 1 female, and no miscarriages. The patient was an only child and as far as is known the patient's mother had had no miscarriages. A perusal of the causes of death in parents and grandparents of the patient reveals nothing besides vascular disorder, although the cause is unknown in some cases. The known forebears died at advanced ages.

In October 1943 she was readmitted with the same complaints which had become more severe in the interim. The patient said that the weakness in the upper extremities had progressed very slowly and that she had noticed muscle fasciculations in the lower lip, hands, and feet. Walking on uneven surfaces had become difficult and climbing stairs virtually impossible. She said that the skin of her hands felt shrivelled and as though it had been immersed in water for a long time. Her son, a physician, pointed out at this time that the patient had been in a car accident in 1924 in which she sustained a severe blow to the top of the head, had not been unconscious, but for some 2 years afterward complained of pain in the back of the neck and between the shoulder blades.

The following additional findings were noted on this admission: fasciculations of the muscles of the dorsal surface of both hands, tremor of the left arm on maintenance of the upper extremities in extension, a bit more flaring of the left scapula than of the right, absence of all deep reflexes in the upper extremities with the exception of the triceps, the left triceps being considerably more active than the right.

Between 1943 and 1947 there were perhaps half a dozen dramatic remissions of the disease in which many, but by no means all, of the symptoms cleared. Two of these remissions occurred immediately after bouts of diarrhea, another after the death of her husband, but the general trend was downhill. Exacerbations could in some instances be correlated with problems arising within the family.

She was again admitted in April 1947. Weakness was now quite apparent in the lower extremities, and all of the deep reflexes both in the upper and lower limbs were absent. Cramps were frequent and severe in the hands and forearms. A psychiatric consultant noted the following during this period in the hospital; the patient had had trouble walking since her late teens, she ascribed this to a knee injury; father was said to have died of tabes dorsalis; at her age 15 or 16 a physician examining her father failed to elicit his knee jerks and in order to demonstrate what the normal reaction should be tapped the patient's patellar tendons and found her knee reflexes to be absent also; according to the patient's memory of her mother, she was a chronically neurotic person who was weak and frail and for whom the patient cared when she took to bed. According to the patient's son, a physician, his maternal grandmother was far from frail, in fact she was to him a dominant personality. He could not remember her ever having seen a phvsician until her final illness with an abdominal aneurysm, the cause of her death at the age of 77 or 78. The patient voluntarily went to sleep before the birth of her twins (her first pregnancy) and did not awaken until after delivery, she required no anesthesia; the patient related attacks of muscle weakness to periods of mental stress; she exhibited "la belle indifference" to her disease; and the patient was unable to squeeze the hand of the examiner but grimaced and obviously avoided closing her hands; she was, however, able to wield a reflex hammer with good strength.

A muscle biopsy from one of the extensors of the wrist (extensor digitorum communis) was performed during this admission and was reported as showing a normal histological picture. This muscle was chosen because of its frequent fasciculation. Free hydrochloric acid was found in the initial specimen of a gastric analysis.

Another short hospital stay occurred in May 1948 following a possible coronary occlusion. She made a good recovery on the customary regimen. Electrocardiograms did not substantiate the diagnosis. She was readmitted in August 1949 for physiotherapy to her knees which had become deformed from osteoarthritis. There was a short period in the hos-

pital in May 1950 for an episode of cramping abdominal pain. Four stool examinations were negative for ova and parasites. Urinalysis and gastrointestinal roentgen series were normal.

In October 1950 neurological examination revealed the following: the only voluntary movements that remained were those innervated by the cranial nerves, and these were within the range of normal. There was slow horizontal nystagmus on attempting to fix the eyes to the right or left. She was unable to swallow much besides liquid; her voice was husky. There was an extremely active snout reflex, and the jaw jerk was not obtained. She was unable to move the limbs or trunk in the slightest. All muscles in the hands showed obvious atrophy, and the palms of the hands were extremely soft. All musculature of the limbs was soft and flabby; atrophy was not apparent in the limbs perhaps because of the liberal overlay of subcutaneous tissue. There was hypotonia in all of the limbs and some pain on manipulation of the joints beyond very slight range. No sensory deficit was found. The superficial abdominal reflexes were unobtainable, and the only deep reflexes elicited were the radials. There was no response to plantar stimulation.

During her final hospital stay she still was obese. She developed episodes of weakness of the respiratory muscles and had great difficulty at times in clearing her throat and trachea of mucous. She gradually grew weaker and immediately after suctioning of the nasopharynx and hypopharynx at 10 P.M. on April 24, 1951 she told her nurse she was about to die, rolled over in bed, and made good her prediction. Heart beat and respirations had ceased when the resident arrived.

She had had numerous and repeated laboratory studies of many sorts including all the serum electrolytes, cholesterol, basal metabolic rate, and urinary porphyrins, none of them revealing any abnormality. Myelography was considered but was not thought warranted by a neurosurgical consultant. All sorts of medicaments were tried without avail including prostigmine, thyroid, glycine, choline, potassium, vitamins, and liver extract. Quinine was given for muscle cramps with relief but it was discontinued when her weakness became worse.

Differential Diagnosis

Dr. Milton Rosenbaum: When Dr. Aring distributes the protocols for the neurological clinical pathological conferences to the students, they are told that they may read whatever they like in preparation for their written discussions. They may discuss the case with fellow students and teachers or with anyone else, since this is the method we follow when faced with a difficult medical problem. So I must tell you that I have pursued the same course in studying this fascinating problem. First, like the students, I went to the books; I read Wilson particularly. Then while attending the meetings of the American Neurological Association I handed around this protocol to various neurologists and their diagnoses varied from amyotrophic lateral sclerosis, which was favored, to cord tumor, but most of them said they really didn't know the diagnosis. So we can all take comfort in the fact that the leading neurologists in the country couldn't give much help with this case. I have had another advantage; it is mentioned in the protocol that a psychiatrist saw the patient. Well, I was the psychiatrist who made the diagnosis of hysteria. As it turns out, I probably came as close to the correct diagnosis as anyone. I will develop this point later.

In certain exercises there is an advantage in not having seen the patient, but with a problem such as this, I believe that I am helped in that I had the "feel" of the patient which it is impossible to derive from a protocol. What actually happened in this case illustrates an interesting point; when handling a patient over a long period of time the physician may become fixated on a certain syndrome and be blinded to other possibilities. In a clinical pathological conference this block can be avoided. The case can be reviewed in broad perspective and the development of an illness viewed in its historical setting.

Now to the protocol. At the age of 64 this woman had had a Pott's fracture, and it was recorded that she cried and screamed for some 24 hours. What is the most important aspect of this little bit of information? Her behavior gives us an idea as to how she reacted to pain and to illness; certainly her behavior was unusual. That is one aspect. The next question is, why did she have a Pott's fracture? To this there is no clue whatsoever. We don't know why or how she fell, but we do know that later she was having difficulty in walking. So it may be that a Pott's fracture before neurological signs attracted the attention of her doctors, was an indication that she was not functioning correctly. I believe there is some evidence that this woman was in trouble for a long, long time.

At the age of 65 there occurred her first admission to the hospital for neurological complaints. For 2½ weeks she had noticed weakness of the upper extremities, with some numbness sounding a bit like root distribution in the left arm, although there were no objective sensory findings. At times people complain of numbness when as a matter of fact they are weak and vice versa. On phys-

ical examination there was weakness of the upper extremities, slight atrophy of the thenar and hypothenar eminences of the hands, and very slight weakness of the lower extremities. There was a note about some flabbiness of the muscles of the upper extremities, an adjective that creeps into the protocol repeatedly as if there was something most strange about this woman's muscles. The objective neurological findings were not too striking except for absent reflexes in the lower extremities. Cerebrospinal fluid was normal. The blood was normal save for slight lymphocytosis and eosinophilia of 6.4 per cent.

If I were her physician at that time, what diagnostic possibilities should I entertain? I would have considered cervical cord tumor, ruptured cervical disc and syringomyelia. With those diagnostic possibilities I would not have been able to explain absence of reflexes in the lower extremities. If she had a cervical disc or a cord tumor, one would wonder how that would effect the lower extremities. Perhaps syringomyelia might account for the signs; there may have been multiple cavities, one in the cervical region of the cord and another in the lumbar region. I would entertain this type of diagnostic thinking because the way I approach neurological cases is to be reasonably certain that I don't miss something that might be remedied, such as a tumor or ruptured intervertebral disc or syphilis. When it comes to the fancier diagnoses—and I believe this patient had some type of fancy disease—there is usually little that we can do therapeutically and then it becomes a sort of academic exercise or narcissistic gratification in making a diagnosis. However, at times the doctor can save the patient time, effort and money by making the correct diagnosis even though little can be done in the way of cure.

The eosinophilia attracted my attention. Now I am rather sensitive to high eosinophile counts, and I think that unless hematology has changed since I went to medical school that 6 per cent is high. The reason for my sensitivity is that the first paper I ever wrote and presented was on "The Neurological Complications of Trichinosis." I could recite it right now because I knew it forward and backward. In looking up the literature as preparation for that paper I learned that a high eosinophile count in a neurologic disorder should make one think of trichinosis, which I think we can rule out here. Dermatomyositis and periarteritis nodosa will have to come up for further discussion.

She was readmitted to the hospital two years later and at that time there were fasciculations, difficulty in walking, and she complained that her hands felt shrivelled as though they had been immersed in water for a considerable length of time. I must admit that when I first went over the case I interpreted this complaint as evidence of some type of sensory disturbance, but on further reflection I decided it might be a clue that she had some primary disease of her muscular system which also affected the skin.

Then there was a bit of additional history —a head injury in 1924 followed by complaints of pain in the back of the neck. Cervical spine injury concomitant with an injury to the head is not a rarity, and changes in the cervical spinal cord may also occur. However, I do not think that the old head injury is important in this case.

The increase of muscle fasciculations brings up another diagnostic possibility namely amyotrophic lateral sclerosis, and I consider progressive muscular atrophy a part of the same syndrome. Certainly amyotrophic lateral sclerosis must have been considered at that time, but against it was the lack of pyramidal tract findings.

The possibility of an intrinsic cord tumor was still present. I say intrinsic because if there were a tumor outside the cord and within the spinal column we would expect root pains which were not experienced. However, I did wonder why a myelogram wasn't done at that time. The probabilities are that her physicians considered it, but may have refrained from carrying it out because of her age, the length of the illness and the remissions and exacerbations. The probabilities are that a diagnosis of amyotrophic lateral sclerosis was entertained at that time.

Then between 1943 and 1947 there were dramatic remissions, which would tend to rule against such diseases as progressive muscular atrophy and amyotrophic lateral

sclerosis, but not against ruptured cervical disc or even a cord tumor. One would think of multiple sclerosis but dismiss it because her symptoms were so uniformly distributed. Other neurological diseases characterized by remissions and exacerbations such as dermatomyositis, periarteritis nodosa, and porphyria would be possibilities. Too often we accept remissions and exacerbations as being spontaneous. I believe that this case gives some hint that emotional factors may have a good deal to do with remissions and exacerbations, regardless of the nature of the underlying disease.

Then in April of 1947, six years after the clinical onset, she again came to the hospital. At this time weakness of the lower extremities was more pronounced, deep reflexes were now lost in both upper and lower extremities and a new symptom had appeared. She complained of cramps. This symptom is seen in patients with neurologic disease especially pyramidal tract disease. However, this patient had no other evidence of pyramidal tract disease, and therefore, my guess would be that the cramps were due to a basic muscular disorder.

During that admission I saw the patient. First I learned that she had trouble walking since her late teens. Then she told me the remarkable story about her father who was supposed to have had tabes dorsalis. He had trouble walking and had lost his knee jerks. When she was a girl of 14 or 15 years, the doctor came to examine her father and pounded on his patellar tendons obtaining no response. Then to illustrate to him what a healthy knee kick should be, the doctor tapped her patellar tendons and there was no response. She also related that her mother was a chronically neurotic person who often took to her bed at which time the patient tended to her. I thought this was interesting in light of the fact that the same thing was happening to her. Our patient would be in bed off and on and had to be cared for. The incident about the pregnancy I considered to be on some sort of an hysterical basis. I made a diagnosis of hysteria. Although I now believe that that was not the whole story, it is rather interesting that all the other neurologists either were insecure or they thought I was pretty good, because from then on the one consistent diagnosis in the records until she died was conversion hysteria. So I had a remarkable influence on the course of ther illness as far as the diagnosis was concerned and perhaps also as far as the treatment. There is one thing that I do want to emphasize. I have enough confidence in my neurological ability to know that I wouldn't have made that diagnosis if there had been any sound evidence of a neurological disorder. I wrote on her chart that the disorder did not resemble any neurological disease that I had ever seen. I may be right there, but as you know, that's not a good way to make a diagnosis. However, I'm certain that she didn't have any of the commoner varieties of neurologic disease such as amyotrophic lateral sclerosis because I don't think that I would have missed them.

As I look back now though, I would review the historical data that I gathered in a different light. I saw her in 1947 when she was 67 years old. Therefore, 50 years had passed since her father was supposed to have had tabes. The question arises, did her father really have tabes? My guess is that the father did not have tabes, rather he had a type of peculiar, rare, interesting neurological disorder, somewhat similar to that of the daughter. It seems likely that she did not have knee reflexes as a child. It could also be so that she had always had some mild difficulty in walking, but for one reason or another it did not bother her until her life situation changed. Certain factors, physical and emotional, might have actually precipitated a disorder that had been lying dormant for a long time. Although the mother was said to have been neurotic and took to her bed, I would also question whether the mother might have suffered from some type of familial muscular neurological disorder. I believe there is enough evidence from the history that I gathered, to make us seriously consider whether we may be dealing with some familial, congenital, or rare type of neurological disease which affected the muscular system.

In 1948 she was admitted for heart complaints which were considered to be possibly a coronary attack, she had an attack of cramping abdominal pain, and osteoarthritis of the knee. One cannot help but wonder

whether we are dealing with a diffuse systemic disease of some sort, if so I would think of dermatomyositis, porphyria, and periarteritis nodosa. If I remember correctly some of her remissions and exacerbations were quite dramatic. I understand she was never well but under certain circumstances she was able to get up and walk after having been confined to bed as a completely helpless invalid. This is why I previously said I was not so far off when I made a diagnosis of hysteria. If I had said she had a functional disease in the true meaning of the term, that is, a disturbance in function without or with little indication of structural change, I think I would have been right.

She was examined again in October 1950 and at this time there were new findings. There was nystagmus on lateral deviation of the eyes. On first reading the protocol and noting nystagmus, difficulty in taking and difficulty in swallowing, I assumed that she had amyotrophic lateral sclerosis or progressive muscular atrophy with bulbar involvement. I do not think so now, and I believe that nothing will be found in the brain stem to account for these signs. If these were signs of neural bulbar palsy the tongue musculature would have been involved and there was no mention of this. I would view the trouble with swallowing and with her voice to be based not on any primary disease in the brain stem but rather as an indication of end-organ involvement, that is of the muscles themselves. Thus again there is evidence of some primary dysfunction in her muscular system. I believe her respiratory death to be on this basis.

In the examination of October 1950 it was again noted that the muscles were soft and flabby. This finding impressed me a great deal and it was one of the reasons that I didn't think she had amyotrophic lateral sclerosis. She was obese. Actually one has to stretch the imagination a great deal to talk about atrophy in our patient, such as is found in the amyotrophies. However, she had had a few muscle fasciculations in 1943. As soon as fasciculations are mentioned to the physician or medical student they immediately think of anterior horn cell disease. It is not widely known that fasciculations may also be seen in patients with no evidence of neurological disease. Dr. Aring[1] and I have seen several physicians who were concerned because they noted muscle fasciculations and there was no evidence of neurological disease. In these and other cases the fasciculations represent an emotional disorder expressed through an increase in muscle tension. It is known that fasciculations may occur in certain types of primary muscle disease, in which case the fasciculations and soft flabby muscles point to a primary disturbance of muscles rather than to disease of anterior horn cell.

I would sum up the diagnostic possibilities by ruling out amyotrophic lateral sclerosis and syringomyelia, cord tumor and ruptured cervical disc. The absence of sensory and pyramidal tract findings and of urinary incontinence pretty well rules out spinal cord disease. However, the loss of deep reflexes and flaccid paralysis points to disease of the neuromuscular system distal to the spinal cord, such as in nerve roots, peripheral nerves, neuromuscular junction or in the muscles themselves. If the history that I myself obtained is worth anything it indicates that this patient suffered for a long time from the disease with which she died. There is a congenital, familial note in this picture, and the main question in my mind is whether she had a truly functional disease. When I say functional I don't mean hysteria, but rather a disturbance of the enzyme systems, affecting muscles or the neuromuscular system at the periphery. I am quite sure that nothing will be found in the central nervous system, that if there are some changes in the anterior horn cells they will not be primary but rather secondary to disuse of muscles. In essence then we have a disease of the motor system, outside of the central nervous system, primarily involving muscle. In favor of this diagnosis are the weakness, the flabbiness, the occasional fibrillations, the cramps and the absent reflexes. There is also evidence of disease in other systems. She suffered from arthritis, cardiac symptoms, acute abdominal symptoms, and she had an eosinophilia. Now what diseases could produce this total picture? There are two possibilities. One would be the so-called collagen diseases, and of

these I think of periarteritis nodosa or dermatomyositis. I went through several textbooks and read numerous articles and found amazing similarity between the story of our patient and some patients with dermatomyositis, although her age and the absence of skin findings are against dermatomyositis unless this might be dermatomyositis without the dermatitis. The abdominal pains, absent reflexes, and muscular findings bring up the possibility of porphyria, but we would have to say porphyria was ruled out by urinalysis. Also in favor of a collagen disease would be the remissions and exacerbations.

Finally I wish to comment on the remissions and exacerbations. The textbooks mention that remissions and exacerbations occur in periarteritis nodosa and dermatomyositis, but they don't tell us why. Our case was studied from a comprehensive point of view and there was evidence that several of the rather dramatic remissions and exacerbations occurred in relationship to certain specific and meaningful emotional situations. We recently studied a patient with dermatomyositis who was transferred from the medical ward to our psychosomatic unit, whose remissions and exacerbations were quite clearly related to certain disturbing factors in his life situation. Emotional factors can influence the course of many diseases and any disease characterized by remissions and exacerbations is fertile field for psychosomatic study. I am not saying that emotional factors caused the disease in the case for today, but I believe they played an important role in the course of her disease, which, as I previously mentioned must have been present for a long time in latent form. It may be one of the collagen disorders is operating here but I imagine the safest diagnosis would be a familial muscular dystrophy of an unclassified type. I favor some primary disease of muscles due to a disturbance in metabolism because of inherent trouble in muscle enzyme systems on a congenital or familial basis.

Dr. Charles D. Aring: The students came to other conclusions in their written discussions; 26 favored the diagnosis of progressive muscular atrophy, 1 amyotrophic lateral sclerosis, 1 taboparesis, 1 that this was an emotional disturbance primarily which led to muscle disuse and atrophy.

Dr. J. Park Biehl: Have you ever seen fasciculations in muscle dystrophy?

Dr. Rosenbaum: I haven't seen too many cases of muscular dystrophy and I don't remember observing muscle fasciculations in them. Fasciculation is not a component of the classical syndrome of progressive muscular dystrophy. The case for today is surely not a classical example of muscular dystrophy.

Discussion of Pathology

Dr. Charles D. Aring: In the first place someone erred badly in reporting as normal the muscle obtained by biopsy in 1947. I have re-examined this material and have had additional sections made, and this muscle is patently disordered as may be seen by reference to figure 1. I am completely at a loss to account for the normal report on the biopsied muscle; in any case this error made for an interesting clinical pathological conference.

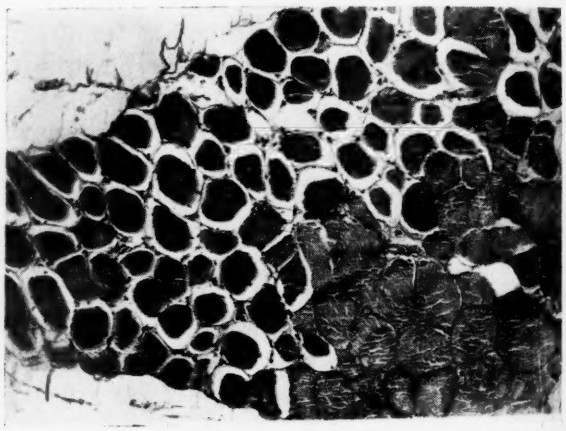

Figure 1. Biopsy of extensor digitorum communis muscle, cross section X 160. Note variance in diameter and in depth of staining of muscle fibres, and retraction of myofibrillary substance from the periphery of many of the muscle fibres. Other fibres appear swollen and granular. Increase in number of sarcolemmal nuclei can be seen in the sheaths of some of the retracted fibres. Hematoxylin and eosin.

In the biopsy, alternating with normal fibres were swollen fibres which took the stain poorly and had lost their cross striations. Hyaline degeneration and breaking up of fibres were found with fair frequency. A few fibres, perhaps 5 per cent of the total, had completely deteriorated and their posi-

tion was marked by a few fine fibrous strands containing numerous sarcolemmal nuclei.

In some areas of this muscle biopsy, irregular staining of the muscle fibres was obvious and appeared to be correlated with the varying size of these fibres. The larger fibres were the lighter stained. Some of these fibres were swollen and granular, with loss of striation, and in these areas there was trouble differentiating individual fibres in longitudinal section. An occasional fibre was divided along its course. In some fibres the nuclei were strung along the center. There was generally an increase in the number of sarcolemmal nuclei.

Occasional fibres would show a massive dilation along its course with little that was stainable in the center; strings of nuclei along the periphery outlined the longitudinal course of the fibre. Certain fibres ran out into a swollen, granular mass which stained less well. Where the damage appeared greatest there had occurred deposits of fat, and in some fascicles there was increased interstitial tissue containing strands of sarcolemmal nuclei.

In cross section of the biopsied muscle, variance in fibre size was apparent (Figure 1) affected areas. There had occurred withdrawal of the myofibrillary substance from the periphery of some fibres to the center (Figure 1). These shrunken fibres stained darker with hematoxulin and eosin, hematoxulin van Gieson, and phosphotungstic acid stains. There was no vascular change in these biopsy sections.

Autopsy was performed 9 hours after death, the body having been embalmed previously through a right inguinal arterial injection. The body measured 158 cm. in length and was estimated to weigh 145 pounds. There was some atrophy of the tissues of the extremities but there was good maintenance of the subcutaneous tissue. The panniculus of fat measured up to 2 cm. The muscle was soft, flabby and pale pink.

Microscopically the lungs showed inflammation of the trachea and bronchi with a few areas of extension into alveoli. There was mild emphysema. There was a moderate degree of generalized arteriosclerosis. In the heart there was focal myocardial fibrosis and patchy fatty vacuolization of the muscle fibres of the left ventricle. In the right ventricle there was some fatty replacement of muscle and one tiny focus of inflammatory cells in the interstitial tissues. There was a leiomyoma of the uterus measuring 4 cm. in diameter. The descending sigmoid colon contained numerous diverticula measuring up to 1 cm. in diameter. There was a simple adenoma in a small nodule in the thyroid gland.

The brain weighed 1350 gm. Gross and microscopic examination (cell, neurofibril and myelin sheath stains) of the brain and spinal cord revealed no abnormalities. The vessels of the circle of Willis were generally thickened but there were no calcareous deposits. Sections from all levels of the spinal cord were normal. There was some fat in anterior horn cells but their constituents were otherwise in good order. Anterior and posterior roots were intact.

At autopsy the muscles were pale and flabby. Microscopically all sections of muscle from many sites showed severe degenerative changes. All of the changes described in the biopsy specimen of muscle were found in accentuated form in the muscles after death. Irregularity of size of muscle fibres with loss of cross striation was characteristic, and many fibres were loaded with chains and clusters of nuclei. Swollen, granular fibres ("hyalin degeneration") in some areas were interspersed between normal appearing fibres. Occasional areas of phago-

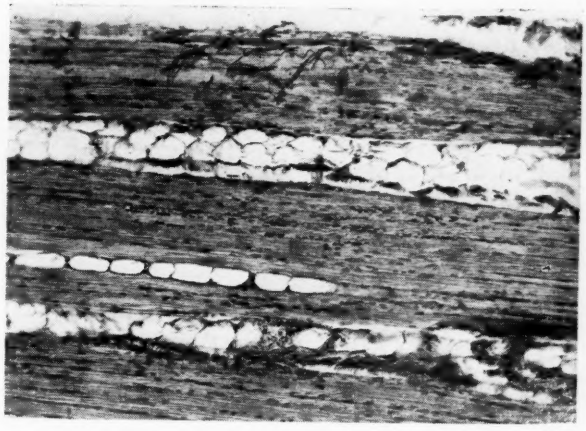

Figure 2. Longitudinal section of a pelvic muscle X 160, taken at autopsy. To show increase in number of sarcolemmal nuclei. There is varying width of fibres and an increase in fat between the fibres. Just below center there is a degenerated fibre whose transition into a chain of fat globules can be clearly seen. Hematoxylin and van Gieson.

cytic response about a fibre in the process of breaking down was seen. Some fibres were replaced by chains of fat globules, and even more striking were those fibres which had maintained some structures and ran out into strings of fat. (Figure 2.) The fat globules were outlined by sarcolemmal nuclei. Increase in sarcolemmal nuclei was also striking.

We can now try to reconstruct the case and come to a diagnostic conclusion. Our problem concerns a woman whose muscular disorder had been followed clinically for a decade up to the time of her death. The clinical onset had occurred at the age of 65 years, and the only example of muscle disorder that can be found in the family was in her father who, it was said, had tabes dorsalis. There is evidence of absent knee reflexes in the patient when she was a girl. Of interest in our patient was her fluctuation in performance during the 7 or 8 years of her disease, from 1941 to 1948, related by the patient herself to mental stress and strain. This fluctuation of great degree was one of the features of her case that led to a considered diagnosis of conversion hysteria.

We find nothing in the tissue studies to explain her disorder besides severe degenerative changes in the muscles. I believe this cas may be considered as an example of late progressive muscular dystrophy. Nevin[2] has given us an excellent description of muscular degeneration in late adult life. In 1936 from the National Hospital, Queen Square, London, he reported two women with primary muscular degeneration with onset at the ages of 57 and 59 years. His search of the literature yielded but 16 cases in which the diagnosis of late progressive muscular dystrophy seemed warranted.

Any other diagnosis seems untenable in our patient. Myositis or dermatomyositis might be thought of, but promptly dismissed since there were no inflammatory changes or relics thereof. Also against this diagnosis was the duration of the disorder. Contracture, so prominent in myositis, was completely absent, a characteristic of late muscular dystrophy which was remarked on by Nevin. At no time had there been lesions of the skin such as are seen in dermatomyositis.

The familial record in sporadic cases in the literature is usually as vague as it was in our case, where there is occasionally mention of a case of "paralysis" in a forebear. However, there are on record families in which there were several cases of myopathy manifesting clinical symptoms first between the ages of 35 and 50 years.

Clinical Diagnosis

Progressive muscular atrophy.

Dr. Rosenbaum's Diagnosis

Progressive muscular dystrophy.

Anatomical Diagnosis

Late progressive muscular dystrophy.
Acute tracheobronchitis.
Early lobular pneumonia.
Diverticulosis of descending colon.

Dr. Daniel Osher: It seems that there are cases of inherited muscle dystrophy, the familial types, in which certain tissues of the body undergo degenerative changes early in life, they don't last out the usual life span. Then there is acquired muscular dystrophy such as I believe this case to be. Apparently acquired muscular dystrophy can be caused by many factors, there may be a relationship to the menopause, or to pregnancy, or to thyroid disease; it appears to be a matter of muscles responding in a limited way to a variety of possible metabolic interferences with their function. I have seen several cases recently that fall into this category of muscular dystrophy, not of classical type; they are important to think about because the disorder is probably amenable if we can find the therapy. The strongly familial muscular dystrophies seem on the less hopeful side from the standpoint of therapy.

Dr. Richard W. Vilter: I would think there would be no particular difference between the muscular dystrophies in families and the more occasional spontaneous occurrences. I followed this patient for years, and I would feel as Doctor Rosenbaum did that something was wrong with the enzyme systems in her muscles. Such disorder whatever it be, could pertain just as well in an acquired as in a congenital variety.

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